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Preventing Relapse after Incentivized Choice Treatment: A Laboratory Model

Mark E. Bouton, Eric A. Thraillkill, Cecilia L. Bergeria, and Danielle R. Davis

University of Vermont

Abstract

Two experiments with rats examined relapse of an operant behavior that occurred after the behavior was suppressed by reinforcing (incentivizing) an alternative behavior. In the first phase, a target response (R1) was reinforced. In a treatment phase, R1 was still reinforced, but a new response (R2) was introduced and associated with a larger reinforcer. As in human contingency management treatments, incentivizing R2 this way was effective at suppressing R1. However, when R2's reinforcement was discontinued, there was a robust and immediate relapse to R1. Experiment 1 found that the strength of R1 during relapse testing was not different from that seen in a no treatment control. Experiment 2 found that relapse could nevertheless be reduced by presenting reinforcers not contingent on responding during the test. Either the reinforcer for R1 or the reinforcer for R2 (which were qualitatively different types of food pellets) were effective. The experiments introduce a laboratory method for studying relapse and how to prevent it after contingency management treatments, and suggest at least one treatment that discourages relapse. The incentivized choice paradigm differs from other models of relapse of operant behavior (e.g., resurgence, renewal, reinstatement) in that it does not focus on the return of behaviors that are inhibited by extinction.

Keywords

relapse; contingency management; resurgence; incentivized choice; extinction

1. Introduction

Unhealthy behaviors such as drug taking, smoking, overeating, and lack of physical activity have become an increasing burden on society. An estimated 40% of premature deaths in the US have been attributed to health-related behaviors (e.g., Schroeder, 2007), and the US lags behind other countries in addressing them (e.g., Bickel, Moody, & Higgins, 2016). The role played by behavior in causing premature deaths may be even greater when considering the recent opioid-overdose epidemic (e.g., Kolodny et al., 2015). Therefore, developing effective methods for encouraging behavior change has become a priority. One method for reducing undesirable behaviors, and encouraging healthier ones, is a treatment known as “contingency management” (CM) (e.g., Fisher, Green, Calvert, & Glasgow, 2004; Higgins, Sigmon, &

Heil, 2011). In contingency management procedures, patients earn reinforcers such as vouchers redeemable for goods or services for not engaging in unwanted behaviors and performing healthier ones (e.g. abstinence from drug taking, medication adherence, attending drug counseling, weight management). Although these treatments are successful at reducing a variety of unwanted behaviors, relapse can occur when reinforcement is discontinued (e.g., Davis, Kurti, Skelly, Redner, White, & Higgins, 2016; Higgins, Silverman, & Heil, 2008). One method of preventing relapse is to continue to maintain new reinforcement contingencies. A type of CM treatment known as the “therapeutic workplace,” where availability of paid work is contingent on abstinence from a target behavior is a viable way to maintain reinforcement contingencies long-term (e.g., Silverman, Svikis, Robles, Stitzer, & Bigelow, 2001). However, this sort of treatment might not be practical for all interventions, and the prevention of relapse remains a critical issue.

The problem of relapse is ubiquitous and is generally consistent with the view that behavioral treatments that encourage new learning do not necessarily erase the old (e.g., Bouton, 2014). For example, extinction is a fundamental behavior-change method in which operant or respondent behaviors are reduced by eliminating the reinforcers that maintain them. After extinction, a number of well-known relapse effects occur that indicate that much of the original learning survives extinction and can return after some manipulation (e.g., Bouton, 2004, 2017). One example is the *renewal effect*, in which context change after extinction causes the extinguished behavior to return (e.g., Bouton & Bolles, 1979; Bouton, Todd, Vurbic, & Winterbauer, 2011; Crombag & Shaham, 2002; Nakajima, Tanaka, Urushihara, & Imada, 2000; Todd, 2013). Renewal is not restricted to extinction. For example, it also occurs when classically-conditioned (respondent) behavior has been reduced by pairing the CS with an oppositely-valenced US (e.g., Holmes, Leung, & Westbrook, 2016; Peck & Bouton, 1990) or when instrumental (operant) behaviors have been suppressed by punishment (Bouton & Schepers, 2015; Marchant, Khuc, Pickens, Bonci, & Shaham, 2013). One conclusion may be that original learning is not erased by behavior change procedures, but may always be available to induce relapse after behavior change (e.g., Bouton, 1993, 2014). One approach is that treatments like extinction and punishment create new inhibitory learning that is especially sensitive to its context for expression (e.g., Bouton, 1993, 2014). This perspective suggests that treatments that increase generalization from the treatment context to potential relapse contexts may be an effective way to prevent relapse.

The perspective is also consistent with recent research on *resurgence*, another relapse paradigm that is thought to have parallels with CM. In studies of resurgence (see Table 1), a target response (R1) is first associated with a reinforcing outcome. In a treatment phase, R1 is then no longer reinforced (it is extinguished), and a new alternative behavior (R2) is introduced and reinforced to replace it (the procedure is often known as *differential reinforcement of alternative behavior*, or DRA). In a final test phase, reinforcement of R2 is discontinued. Although both R1 and R2 are now extinguished, the typical finding is that some recovery of R1 occurs (it “resurges”) (e.g., Leitenberg, Rawson, & Bath, 1970; Liewing & Lattal, 2003; Winterbauer & Bouton, 2010; see Craig, Nall, Madden, & Shahan, 2016, and Podlesnik, Jimenez-Gomez, & Shahan, 2006, for examples of resurgence after the

extinction of drug seeking). Like renewal (and other relapse paradigms), resurgence indicates that extinction of R1 does not create full erasure or unlearning.

One explanation of resurgence is that it is a new form of the familiar renewal effect (e.g., Winterbauer & Bouton, 2010). That is, when reinforcement of R2 is discontinued, the reinforcement context changes, and the extinguished response recovers, just as it does with other types of context change (e.g., Bouton et al., 2011; Todd, 2013). This view suggests that treatments that encourage generalization between the contexts of treatment and relapse testing will reduce the resurgence effect. A number of findings are consistent with this idea. For example, alternating sessions of R2 reinforcement and R2 extinction during treatment reduces resurgence markedly (Schepers & Bouton, 2015). (Extinction sessions are identical to resurgence test sessions; thus, extinction of R1 during them would be expected to generalize to resurgence testing.) In another example, Bouton and Trask (2016) reinforced R1 with a specific outcome (O1) in the first phase; in the second, while R1 was extinguished, R2 was reinforced with a different outcome (O2). In a final test, when reinforcement of R2 was discontinued, R1 resurged. However, if O2 was presented freely (not contingent on responding) during testing, it eliminated the resurgence effect. In contrast, noncontingent presentations of O1 did not. Such findings are consistent with the idea that R1 inhibition learned during extinction (e.g., Bouton, Trask, Carranza-Jasso, 2016; Rescorla, 1997) was specific to the context provided by presentations of O2. When that context was maintained during testing, extinction performance (behavioral inhibition) was maintained. These findings, and others, are not anticipated by other views of resurgence (e.g., Shahan & Sweeney, 2011; Shahan & Craig, 2017), but are consistent with the context renewal view (see Trask, Schepers, & Bouton, 2015, for a more extensive review of the context view).

Although resurgence bears some resemblance to relapse after CM treatments, the paradigm was not designed as a model of CM, and there are consequently a number of differences between them (e.g., see Bouton & Schepers, 2014). Perhaps the most significant difference is that the target behavior is not typically extinguished in CM. That is, if a drug user, smoker, or overeater happens to commit the target behavior during treatment, that behavior will almost certainly be reinforced. Unlike resurgence, CM is not an extinction paradigm. The main purpose of the present article was therefore to develop a new model of relapse that respects this feature of CM. Our *incentivized choice paradigm* is sketched in the lower half of Table 1. In this paradigm, as in resurgence, the animal is reinforced for a target behavior (R1) in the first phase. In the subsequent treatment phase, an alternative behavior (R2) is also introduced and reinforced. However, R1 is not extinguished during treatment; it can still earn reinforcers according to the reinforcement schedule used in Phase 1. Instead, R1 is suppressed by merely incentivizing R2; that is, by associating R2 with an even larger (more motivating) reinforcer. In preliminary (unpublished) work, we found that reinforcing R2 with nine 45-mg food pellets (while reinforcing R1 with only one) encouraged a near-exclusive preference for R2. (That result corresponds to evidence in the CM literature that larger-magnitude alternative reinforcers are associated with higher rates of abstinence during treatment [Lussier, Heil, Mongeon, Badger, & Higgins, 2006]). In the final relapse test, R2's reinforcement is then discontinued. Since R1 has been reinforced throughout, the probability that the rat will return to R1 (and exhibit relapse) is expected to be 1.0. However, the

procedure provides a method that can be used to study treatment innovations that might nonetheless slow the relapse down. The present experiments introduce the method and provide a test of one such innovation.

2. Experiment 1

The first experiment involved two groups. One group (Group Incentivized Choice) received a three-phase procedure like the one just described. In Phase 1, R1 (presses on a lever) was reinforced with a single 45-mg food pellet (O1; either a sucrose- or grain-based pellet, counterbalanced). In the second (treatment) phase, R1 was still reinforced, but R2 (presses on a second lever) was introduced and reinforced with 9 pellets per reinforcer (O2; either grain- or sucrose-based pellets, counterbalanced). In the final test, reinforcement of R2 was discontinued. As described above, we expected rapid relapse to R1. To assess carry-over of the Phase-2 incentivized choice treatment, we also included a No Treatment control. This group received the same R1 training in Phase 1, but was only handled on the days during which Group Incentivized Choice received its Phase-2 treatment. During final relapse testing, the No Treatment group's R1 performance allowed an estimate of how well R1 was simply retained over time, without influence of the contingency management treatment in Phase 2.

Several aspects of the procedure are worth noting. First, to preserve the character of CM, the reinforcer for R2 was qualitatively different from the one that reinforced R1. Second, R1 was reinforced on a ratio schedule (rather than an interval schedule). An RR schedule may mimic how unhealthy behaviors, like drug taking, are reinforced and performed at high rates which often monopolize a large proportion of time and effort. In addition, R1's reinforcement rate (like its response rate) could decrease when R2 was introduced and incentivized. (A reduction in reinforcement rate would not necessarily occur if R1 were instead reinforced on an interval schedule.) This feature of the procedure allowed us to study the suppression of O1 reinforcers that were earned during treatment, which is a critical focus of analogous CM treatments. Finally, the alternative behavior (R2) was reinforced according to a random-interval (RI) schedule. Like CM treatments, RI schedules arrange alternative reinforcers after a scheduled amount of time, rather than a specific number of R2 responses.

2.1 Method

2.1.1 Subjects—The subjects were 16 female Wistar rats obtained from Charles River (St. Constance, Quebec). The rats were approximately 85–95 days old at the start of the experiment and were housed individually in suspended stainless steel cages in a room maintained on a 16: 8-hr light: dark cycle. Prior to the beginning of the experiment, all rats were food deprived to 80% of their free-feeding weight and maintained at that level throughout the experiment with a supplemental feeding following the experimental session each day.

2.1.2 Apparatus—Eight operant chambers sized $31.75 \times 24.13 \times 29.21$ cm ($l \times w \times h$) were housed in individual sound-attenuation chambers. The front and back walls of the chambers were aluminum, and the sidewalls and ceiling were clear acrylic plastic. Recessed $5.1 \text{ cm} \times 5.1 \text{ cm}$ food cups with infrared photobeams positioned approximately 1.2 cm

behind the plane of the wall and 1.2 cm above the bottom of the cup were centered in the front wall about 3 cm above the floor. 4.8 cm stainless steel operant levers (Med Associates, Fairfax, VT: ENV-112) were located to the left and to the right of the food cup, 6.2 cm above the floor. Background noise (60 dBA) was provided by ventilation fans, and illumination was provided by two 7.5-W incandescent bulbs mounted on the ceiling of the sound-attenuation chamber. In one set of boxes, the floor consisted of 0.48-cm diameter stainless steel grids spaced 3.81 cm apart and mounted parallel to the front wall. The apparatus was controlled by computer equipment located in an adjacent room. Although the two sets of boxes can provide different contexts, they were not used in that capacity here. Two reinforcers were used: a 45-mg grain-based pellet (5-TUM; 181156; TestDiet, Richmond, IN, USA), and a 45-mg sucrose pellet (5-TUT; 1811251; TestDiet, Richmond, IN, USA).

2.1.3 Procedure

2.1.3.1 Magazine training.: All rats received two magazine training sessions, one with each type of reinforcer, on the two days prior to beginning Phase 1. With the levers retracted, pellets were delivered during each session on a random time 30-s schedule, resulting in approximately 60 pellets delivered during each session. Pellet type was counterbalanced so that half the rats were trained first with O1 (grain or sucrose, counterbalanced) and the other half were trained first with their O2 pellets (sucrose or grain, counterbalanced).

2.1.3.2 R1 acquisition (Phase 1).: Over the next 12 days, rats received one session each day in which pressing a lever (R1; right or left, counterbalanced) was reinforced with a food pellet (O1; grain or sucrose, counterbalanced). Pellets were delivered according to a random ratio (RR) 2 on days 1–2, RR 5 on days 3–4, RR 15 on days 5–6, RR 20 on days 7–8, and RR 25 on days 9–12. Each session was initiated by the insertion of the lever after 2 min. Sessions ended with the retraction of the lever when the rat had earned 30 pellets.

2.1.3.3 R2 acquisition (Phase 2).: Half the rats (Group Incentivized Choice; $n = 8$) received 8 daily 30-min sessions in which R2 was reinforced with a large magnitude reinforcer. Both the left and right levers were inserted after 2 min and remained in place throughout the session. Presses on the newly inserted lever (R2) were reinforced on a random interval (RI) 30-s schedule with the second type of food pellet (O2; grain or sucrose). Each O2 reinforcer consisted of nine pellets presented in succession (0.4-s interval between pellets). The target lever (R1) remained in the chamber and was reinforced with a single O1 pellet on RR 25. The remaining rats (Group No Treatment; $n = 8$) were handled in the same way as the Incentivized Choice group each day, but were returned to their home cages without exposure to the operant chamber.

2.1.3.4 Test phase.: All rats then received four daily 30-min test sessions in which responses to R2 had no programmed consequences (that is, were extinguished). In contrast, R1 continued to be reinforced with a single O1 pellet according to RR 25.

2.1.4 Data analysis—We focused not only on response rate, but on the rate at which O1 reinforcers were earned. Data were analyzed with JASP statistical software (JASP Team,

2016). Group mean data were analyzed with analyses of variance (ANOVAs) with a rejection criterion of $p < .05$. Effect sizes and Bayes factors (Jeffreys, 1961; Rouder et al., 2016) are reported when relevant to the hypotheses. In some cases, planned comparisons using error terms from the overall ANOVA were used to assess pairwise differences.

2.2 Results

The results are summarized in Figure 1. As shown in Figure 1a, all rats acquired R1 across sessions of Phase 1, and the rate of R1 responding did not differ between groups. A Group (Incentive Treatment, No Treatment) by Session (12) ANOVA found only an effect of Session, $F(11, 154) = 46.53$, $MSE = 88.05$, $p < .001$; other F s < 1 . A simple group comparison also found no difference in the final session of Phase 1, $F(1, 14) = 1.36$, $MSE = 195.70$. The rate of reinforcement for R1 in the final sessions of Phase 1, when the groups had reached RR 25, is shown in Figure 1b. A Group by Session ANOVA found no reliable differences between groups, largest $F(3, 42) = 2.81$, $MSE = 0.34$, and no difference in the final session, $F < 1$.

Figure 1c shows the rates of R1 and R2 responses in Group Incentivized Choice in Phase 2, when R2 was introduced and reinforced. The treatment was effective at reducing R1's rate, as well as the rate of delivery of O1 (Figure 1d). Rats decreased their R1 responding in the first session of Phase 2, relative to the last session of Phase 1, $t(7) = 3.64$, $p = .008$. Response rate on R1 then decreased further over the remaining sessions, $F(7, 48) = 3.39$, $MSE = 85.70$, $p = .005$. Along with decrease in R1 rate, the obtained rate of O1 reinforcement decreased from the last session of Phase 1 to the first session of Phase 2, $t(7) = 4.17$, $p = .004$, and then decreased from there over the remaining sessions of Phase 2, $F(7, 49) = 3.34$, $MSE = 0.21$, $p = .006$. Response rates on R2 increased between sessions 1 and 8, $F(1, 7) = 8.34$, $MSE = 2.24$, $p = .02$. Rate of O2 reinforcer delivery (per min; not shown) also increased from session 1, $M = 0.7$, to session 8, $M = 1.0$, in Phase 2, $t(7) = 3.41$, $p = .01$. Remember that a reinforcement rate of 1.0 meant that the rats were earning nine O2 pellets per minute.

The right side of Figure 1c shows R1 and R2 response rates during the test phase, when reinforcement of R2 was discontinued. As the figure indicates, there was an abrupt return to R1 responding in Group Incentivized Choice at this time. In fact, R1 responding immediately returned to the same rate shown by the no treatment control. Statistical analysis confirmed that response rate in Group Incentivized Choice increased from the final session of Phase 2 to the first session of the test, $t(7) = 6.21$, $p < .001$. (O1 reinforcer rate also increased, $t(7) = 5.98$, $p < .001$.) And when we compared R1 response rates in the two groups over all the sessions of testing in a Group by Session ANOVA, we found only an effect of session, $F(3, 42) = 8.91$, $MSE = 141.91$, $p < .001$; the Group effect and Group $\times$ Session interaction did not approach significance, F s < 1 . Bayes factors supported the null hypothesis of no group difference during the test for response rates, $BF = 0.51$, and reinforcer rates, $BF = 0.53$. R2 responding in Group Incentivized Choice increased during the first session of testing relative to the last session of Phase 2, $t(7) = 3.12$, $p = .02$, but then decreased over the remaining sessions, $F(3, 21) = 30.22$, $MSE = 2.13$, $p < .001$.

To reach a deeper appreciation of the dynamics of relapse to R1, we also examined R1 responding over each minute of the first two sessions of relapse testing. These results are presented in Figure 2. In order to understand how responding in testing compared to initial training, each animal's response rate was expressed as the proportion of the mean response rate that had been achieved during the last session of Phase 1. As the figure indicates, although both the treatment and no treatment groups started at a rate that was lower than that shown at the end of Phase 1, there was no difference between the groups during either session, Group effect $F_s < 1$, Group by Session and Group by Block interaction $F_s < 1$. The results provide little support for the idea that the present incentivized choice treatment suppressed R1 responding at all once the treatment was discontinued.

2.3 Discussion

Reinforcing the alternative behavior (R2) with a large reinforcer was effective at reducing R1, although it did not reduce it to zero. The reduction of R1 also reduced the rate at which O1 reinforcers were earned. Although this is an obvious consequence of the ratio schedule of reinforcement used here, the rate of reinforcement may be a useful dependent variable in studying treatments whose goal is to reduce the intake of reinforcers that are harmful to health (drugs, cigarettes, junk food). However, the main finding of the experiment was that the suppression of R1 (and its rate of reinforcement) was only temporary; when reinforcement of R2 was discontinued, there was a rapid return to R1. Indeed, this relapse to R1 was evidently so rapid that there was no detectable difference between the Incentivized Choice Group and a No Treatment Control. The results are reminiscent of the challenge that confronts CM treatments, which likewise suppress problematic R1 behavior by incentivizing an alternative one.

3. Experiment 2

As noted earlier, although relapse after an incentivized choice treatment may be very rapid, the procedure allows investigation of treatment modifications that might slow that rapid relapse down. The second experiment provided an initial investigation of one such modification. As described in the Introduction, in the extinction-based resurgence paradigm, treatments that increase the similarity between the treatment and relapse phases have been shown to reduce relapse after treatment. This is expected, because extinction performance is especially sensitive to context change (e.g., Bouton & Todd, 2014). Experiment 2 was therefore designed to study the effect of a resurgence-eliminating treatment investigated by Bouton and Trask (2016). Recall that they found that noncontingent presentations of the treatment (Phase 2) reinforcer during relapse testing, but not the original (Phase 1) reinforcer, eliminated the resurgence effect. Experiment 2 used the basic incentivized choice procedure used in Experiment 1: R1 was first reinforced with O1, and during the treatment phase R2 was introduced and reinforced with a large (9-pellet) presentation of O2. In the relapse test, reinforcement for R2 was discontinued. Three groups differed in their treatment during the relapse testing phase. The control group received only extinction of R2; these subjects were expected to relapse to R1, as in Experiment 1. However, a new group (Group O2) continued to receive noncontingent O2 presentations at the same rate at which they had been programmed during treatment. The second new group (Group O1) received a similar

treatment, but with noncontingent presentations of O1 instead of O2. If increasing the similarity between the treatment and relapse phases of the current method is as important as it is in resurgence, then O2 presentations might be especially effective at slowing relapse when R2 reinforcement is discontinued.

Although our previous unpublished work suggested that 9-pellet reinforcers are necessary to suppress R1 responding in the paradigm (they were more effective than 3-pellet or 1-pellet reinforcers), it is possible that relapse might be prevented by reinforcers of a smaller magnitude. To explore the importance of the size of the noncontingent reinforcer presented during testing, we varied the size of the noncontingent reinforcers (both O2 and O1) during testing. Over test sessions 1–4, the number of pellets delivered during each noncontingent reinforcer presentation was 9, 3, 1, and 0, respectively.

3.1 Method

3.1.1 Subjects and Apparatus—The subjects were 32 naïve female Wistar rats obtained from the same supplier and maintained under identical conditions to Experiment 1. The apparatus was the same as that used in Experiment 1.

3.1.2 Procedure—Magazine training and Phase 1 conditioning followed the procedures described in Experiment 1. All rats then received Phase 2 training following the procedure that was given Group Incentivized Choice in Experiment 1. Testing then consisted of four daily 30-min sessions. For all groups, R1 continued to be reinforced, as usual, with one O1 pellet on RR 25. However, R2 was now extinguished. In each session, Group Extinction ($n = 8$) received no pellets for pressing R2. In contrast, Group O1 ($n = 12$) received noncontingent O1 and Group O2 ($n = 12$) received noncontingent O2 pellets according to a variable time 30-s schedule (RT 30 s). The number of pellets presented in succession during presentation of these noncontingent reinforcers was 9, 3, 1, and 0 over the four sessions of the test.

3.2 Results

The results of the experiment are summarized in Figure 3. As before, rats similarly acquired R1 across sessions of Phase 1 (Figure 3a). A Group (O1, O2, Extinction) by Session (12) ANOVA found only an effect of Session, $F(11, 319) = 83.66$, $MSE = 99.70$, $p < .001$, other $F_s < 1$. A simple group comparison on the final session found no difference, $F(2, 29) < 1$. Figure 3b confirms that reinforcement rate (O1 per min) was also similar between groups over the final sessions of Phase 1, when O1 was delivered according to RR 25. A Group by Session ANOVA found an increase in reinforcer rate across these sessions, $F(3, 87) = 4.19$, $MSE = 0.52$, $p = .008$, but no other effects, $F_s < 1$. A simple comparison of reinforcer rate in the final session found no differences between groups, $F < 1$.

The left part of Figure 3c shows responses rates of R1 and R2 in each group over sessions of Phase 2. All groups decreased their R1 responding in the first session of Phase 2 relative to the last of Phase 1, $F(1, 29) = 283.38$, $p < .001$, and then response rate further decreased over the sessions of Phase 2, $F(7, 203) = 3.87$, $MSE = 36.60$, $p < .001$. The group effects and interactions were not reliable, $F_s < 1$. Reinforcer rates for R1 (left portion of Figure 3d) also

decreased from the last session of Phase 1 to the first session of Phase 2, $F(1, 29) = 243.87$, $MSE = 0.56$, $p < .001$, and further decreased over the sessions of Phase 2, $F(7, 203) = 2.79$, $MSE = 0.07$, $p = .009$. There were no group effects or interactions, largest $F = 1.33$. Not surprisingly, the rate of R2 also increased from session 1 to session 8, $F(1, 29) = 6.39$, $MSE = 1.53$, $p = .02$; the group effect and interaction were not significant, $F_s < 1$. Reinforcer (O2) rate for R2 also increased from session 1 ($M_s = 0.6, 0.7$, and 0.6) to session 8 ($M_s = 0.9, 0.9$, and 0.9) in Groups O1, O2 and Extinction, $F(1, 29) = 77.05$, $MSE = 0.02$, $p < .001$; there was no group effect or interaction, largest $F = 2.33$.

As shown on the right side of Figure 3c, R1 responding in Group Extinction rapidly increased from the final session of Phase 2 to the first session of testing, as we saw in the similar group in Experiment 1. However, R1 responding in Groups O1 and O2 did not return as rapidly. A Group by Session (Last Phase 2, First Test) ANOVA found effects of Session, $F(1, 29) = 18.52$, $MSE = 127.00$, $p < .001$, Group, $F(2, 29) = 16.12$, $MSE = 200.40$, $p < .001$, and a reliable Group by Session interaction, $F(2, 29) = 21.64$, $p < .001$. Simple ANOVAs found no difference in responding in the last session of Phase 2, $F < 1$, but there was a significant difference between groups in the first test session, $F(2, 29) = 19.77$, $MSE = 301.70$, $p < .001$. Corrected posthoc tests (Tukey) found that Groups O1 and O2 each differed from Group Extinction, $p_s < .001$, but not from each other, $p = .99$. Over sessions of testing in which the number of O2 pellets decreased, R1 responding increased in Groups O1 and O2. A Group by Session ANOVA found significant effects of Group, $F(2, 29) = 6.77$, $MSE = 2211.00$, $p = .004$, Session, $F(3, 87) = 54.69$, $MSE = 235.60$, $p < .001$, and a Group by Session interaction, $F(6, 87) = 3.03$, $p = .01$. A Bayesian ANOVA suggested support for the null hypotheses of similar R1 rates in Groups O1 and O2 across sessions of testing, $BF = 0.34$. It is worth noting that, in the final session of the test, when R2 was not reinforced and reinforcers were no longer delivered noncontingently in any group, there were no differences among the groups, $F(2, 29) = 1.11$, $MSE = 1330$, $p = 0.34$. The results indicate that noncontingent presentations of O1 and O2 both significantly slowed relapse to R1 observed when R2 reinforcement was otherwise discontinued. Notice that the right part of Figure 3d indicates that the reinforcers earned for R1 followed the same general pattern.

Response rates on R2 showed a complex pattern from the last session of Phase 2 to the first session of the test. There were reliable effects of Session, $F(1, 29) = 5.18$, $MSE = 4.94$, $p = .03$, and a Group by Session interaction, $F(2, 29) = 10.08$, $p < .001$. Follow-up comparisons (Tukey) found that R2 responding in Group O2 ($M = 1.9$) was lower than that in Groups Extinction ($M = 7.7$), $p = .005$. Group O1 ($M = 4.6$) did not differ from either group, $p_s > .17$. Animals reduced their R2 responding across the remaining test sessions; a Group by Session ANOVA revealed a Session effect, $F(3, 87) = 3.96$, $MSE = 5.19$, $p = .01$. Although there was no group effect, $F < 1$, there was a significant Group by Session interaction, $F(6, 87) = 9.67$, $p < .001$. A similar ANOVA that compared the Extinction group with Groups O1 and O2 collapsed also revealed a Group by Session interaction, $F(3, 90) = 13.43$, $MSE = 5.76$, $p < .001$. One effect of delivering the noncontingent reinforcers was apparently to decrease the rate of R2's extinction (e.g., Rescorla & Skucy, 1969; Winterbauer & Bouton, 2011).

As noted above, delivery of O1 or O2 during testing markedly interfered with relapse to R1. One reason might be that delivery of either reinforcer noncontingently could have elicited food cup behavior that interfered with performance of R1. We therefore asked whether time in the food cup was negatively correlated with the level of R1 responding. Figure 4 shows R1 responding as a function of percent time in the food cup for each rat during the sessions in which Groups O1 and O2 received noncontingent reinforcers (test sessions 1–3). As the figure suggests, percentage time in the food cup was not negatively related with R1 responding. For Group Extinction, the correlation between the two measures was $r(6) = +.47$, $p = .24$ (note that the relationship would have been less positive if the single outlier were removed). For Groups O1 and O2 together, the correlation was $r(22) = -.19$, $p = .38$. Food cup behavior accounted for only about 3% of the variation in R1 responding in the latter groups ($r^2 = .03$). The results thus suggest that food cup behavior did not reliably predict the rate of R1 responding, as one would expect if food cup responding merely competed with R1.

3.3 Discussion

As in Experiment 1, reinforcing R2 was effective at reducing R1 and the number of O1 reinforcers that it earned. Also as in Experiment 1, discontinuing reinforcement of R2 after treatment led to a rapid return to R1 responding (Group Extinction). A new result, however, was that presenting reinforcers noncontingently during relapse testing suppressed the relapse to R1. This effect depended on the size of the noncontingent reinforcer presented during testing; as the number of pellets declined over sessions, suppression of R1 also declined. However, another finding was that the identity of the reinforcer delivered noncontingently had no impact on its effectiveness: Presenting either O1 (the reinforcer from Phase 1) or O2 (the reinforcer from Phase 2) suppressed R1. This result suggests that the similarity of the reinforcer delivered during treatment and testing may play less of a role in the incentivized choice paradigm than it does in the extinction-based resurgence paradigm (Bouton & Trask, 2016). The difference may not be surprising, because context change generally has more impact on extinction performance (targeted in resurgence) than acquisition performance (targeted in incentivized choice) (e.g., Bouton & Todd, 2014). Thus, discriminative control by the specific reinforcer delivered in the treatment phase might be expected to have a bigger effect on resurgence than it did here. It is worth noting that the noncontingent reinforcers studied here may be analogous to the natural reinforcers that emerge and accompany a drug-free lifestyle after successful CM treatment. The results may thus imply that such reinforcers might be effective at suppressing unwanted target behavior despite their specific qualitative nature.

There are at least two explanations of how the noncontingent O1 and O2 slowed relapse to R1. One possibility is that delivery of either reinforcer elicited incompatible behaviors that interfered with the rat's ability to perform R1. Although it is difficult to rule this possibility out completely, our analysis of one potentially competing behavior—time in the food cup—suggested that it bore no relation to R1 responding in individual rats. The amount of food cup behavior and the amount of R1 responding were not negatively correlated. Perhaps the slowing of relapse did not depend on mechanical response competition. The effect of adding noncontingent reinforcers is also consistent with models of reinforcement, like the

quantitative law of effect (e.g., Herrnstein, 1970; McDowell, 2013), which emphasize that the rate of R1 will be reduced by reinforcement of “other” behavior. (Although the quantitative law of effect emphasizes “other” behaviors, it does not give a role to response competition.) Interestingly, the present results suggest that the effectiveness of alternative reinforcement at reducing R1 does not depend on the specific stimulus properties (sucrose vs. grain) of a food reinforcer. That is, the sucrose and grain pellets appeared to function as substitutes here (e.g., Green & Freed, 1998).

4. General Discussion

The present experiments suggest that the incentivized choice paradigm may be useful in studying relapse processes that may come into play after contingency management treatments are discontinued. In both experiments, reinforcing an alternative behavior (R2) suppressed a still-reinforced target response (R1), just as it would in a contingency management treatment. However, although reinforcement of R2 clearly suppressed R1 responding, there was no evidence that it prevented relapse of R1 when the treatment was discontinued. Indeed, in Experiment 1, R1 returned so rapidly that we observed no difference between responding in the treatment group and a no treatment control; as noted earlier, that result may underscore the challenge faced by true contingency management therapies. Nonetheless, Experiment 2 established that continuing to present reinforcers noncontingently during the relapse phase slowed the relapse to R1. And importantly, that effect did not depend on the identity of the noncontingent reinforcer; noncontingent presentations of either the reinforcer used in the incentivized treatment (O2) or the reinforcer used to originally maintain the target behavior (O1) were effective at slowing relapse. The results are overall consistent with accounts like the quantitative law of effect (e.g., Herrnstein, 1970; McDowell, 2013): Reinforcing R2 during treatment, and continuing to present reinforcers during relapse, should increase reinforcement of other behavior and consequently reduce the rate of R1. The present sucrose- and grain-based reinforcers were evidently substitutable. As noted earlier, the translational significance of Experiment 2's results may be that natural reinforcers that can be encouraged as a patient becomes drug-free may also serve to suppress unwanted target behavior after CM treatment ends regardless of their specific nature.

The present method bears some relation to other experiments in which target behaviors have been suppressed by incentivizing an alternative response instead of extinguishing them. For example, in an experiment with rats reported by Ginsburg and Lamb (2013), the presence of one discriminative stimulus (SD) signaled that responses on one lever would be reinforced with ethanol on a rich fixed-ratio (FR) 5 schedule and responses on another lever would be reinforced with food on a lean (FR 150) schedule. The rats tended to respond for ethanol over food. In a second SD, the ratio for food seeking was decreased to FR5; after incentivizing the food response this way, food responding came to suppress ethanol responding. However, several sessions of this training did not prevent renewal to ethanol responding when the first SD was reintroduced. In another related method, Caprioli et al. (e.g., 2015) produced “voluntary abstinence” from methamphetamine-seeking by introducing discrete trials in which rats could choose to press different levers for methamphetamine vs. food. When given such choices, the rats chose food, and thus

suppressed their methamphetamine intake. However, voluntary abstinence produced this way for 21 days did not prevent an “incubation of craving;” when returned to trials with the methamphetamine lever only, rats responded more vigorously after 21 days than after 1 day of voluntary abstinence training.

The fact that noncontingent O2 presentations were no more effective than O1 presentations at reducing relapse in the present Experiment 2 contrasts with results in the resurgence paradigm (Bouton & Trask, 2016). As emphasized above, in resurgence, noncontingent presentations of O2 continued to suppress an extinguished R1 after DRA, but O1 presentations did not. The results thus suggest that the variables that control relapse after a DRA (resurgence) procedure may differ from those that control relapse after incentivized choice. One possibility is that discriminative control of behavior by the sensory properties of the reinforcers plays a more important role in the resurgence paradigm. This is consistent with the fact that the resurgence paradigm, but not the incentivized choice paradigm, focuses on relapse of *extinguished* operant behavior. As noted earlier, extinguished responding is known to be especially sensitive to the effects of context change (e.g., Bouton & Todd, 2014). And equally important, the idea that the generalization between Phases 2 (treatment) and 3 (relapse testing) will reduce relapse requires that the organism actually learns something new about R1 during treatment that can generalize between treatment and testing. In extinction, that new learning has been tentatively identified as active inhibition of the response (e.g., Bouton, 2017; Bouton et al., 2016; Rescorla, 1997). Thus, resurgence is reduced when response inhibition generalizes from extinction to the test. In contrast, in the incentivized choice paradigm, there is no evidence that the rat learns anything new about R1 when R2 is introduced and incentivized. Indeed, models that describe choice between R1 and R2 (like the quantitative law of effect) are generally performance models that would not imply any new learning about R1 in the present treatment phase. Thus, incentivizing an alternative response would suppress R1 performance, but not necessarily create any new learning about R1. In contrast, in the resurgence paradigm, it is the new learning about R1 that occurs in extinction that comes under the control of the discriminative properties of the reinforcer and can generalize between extinction and testing (Bouton & Trask, 2016; Trask & Bouton, 2016).

Whatever the value of these theoretical ideas, the present experiments establish a new laboratory preparation that may provide a tool to study treatment variables that might encourage behavioral maintenance, and discourage relapse, when reinforcement is discontinued after contingency management treatment.

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5. References

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Figure 1.

Results of Experiment 1. a.) Acquisition of R1 across sessions of Phase 1. b.) Obtained O1 reinforcer rates across the final four sessions of Phase 1. c.) R1 (top) and R2 (bottom) response rates across Phase 2 and Test. d.) O1 reinforcer rates across Phase 2 and Test. Error bars represent standard error of the mean.

Figure 2.

Finer-grained analysis of R1 responding in each minute of the first two sessions of relapse testing in Experiment 1. Sessions are separated by the vertical dashed line. Data are expressed as the mean proportion of the response rate achieved in the final session of Phase 1. Error bars represent the standard error of the mean.

Figure 3.

Results of Experiment 2. a.) Acquisition of R1 across sessions of Phase 1. b.) Obtained O1 reinforcer rates across the final four sessions of Phase 1. c.) R1 (top) and R2 (bottom) response rates across Phase 2 and Test. d.) O1 reinforcer rates across Phase 2 and Test. Error bars represent standard error of the mean.

Figure 4.

Relationship between R1 response rate and percent of session time spent in the food cup during the Phase 3 (Relapse Test) sessions in which free reinforcers were presented to Groups O1 and O2. Dashed regression line is for Group Extinction; solid regression line is for Groups O1 and O2.

Table 1

Resurgence and Incentivized Choice Paradigms Compared

Phase 1	Treatment (Phase 2)	Relapse Testing
Resurgence Paradigm		
R1→O	R1-	R1-
	R2→O	R2-
Incentivized Choice Paradigm		
R1→O	R1→O	R1→O
	R2→O	R2-

Note: R1 = target response; R2 = alternative response; O = reinforcing outcome; → = caused; - = extinction. In both paradigms, the target response (R1) is suppressed during the treatment phase, but returns (relapses) during relapse testing.